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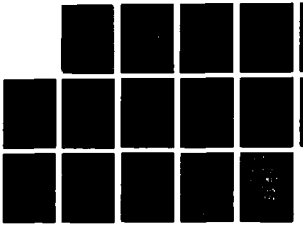

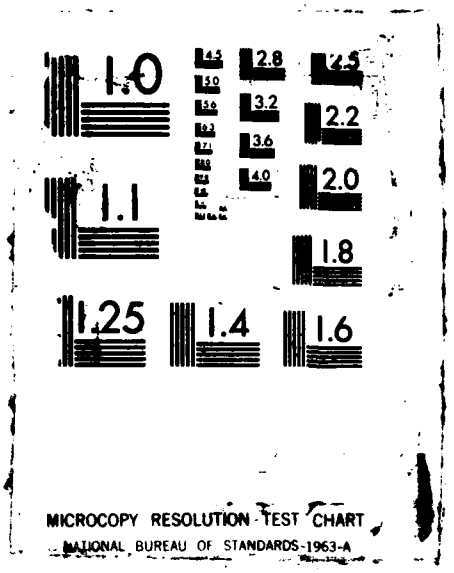
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Temperature Regulation at Rest and Exercise During the Human Menstrual Cycle

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Thermoregulatory responses were studied in eight women during three separate experimental protocols in both the follicular (F) and luteal (L) phases of the menstrual cycle. Continuous measurements of esophageal temperature (T_{es}), mean skin temperature ($\bar{T}_{sk}$), metabolism, and forearm sweating ($\dot{m}_s$) were made during all experiments. Study I evaluated 35 minutes of seated cycle exercise (60% $\dot{V}O_2$ peak, T_a = 35°C, T_{dp} = 14°C). Study II involved both passive heat exposure and seated cycle exercise (80% $\dot{V}O_2$ peak; T_a = 50.0°C, T_{dp} = 18.5°C) to elicit a 0.8°C increase in T_{es}. Study III evaluated high intensity exercise (35 minutes, 80% $\dot{V}O_2$ peak) at T_a = 35°C and T_{dp} = 10°C. The normal L increase in resting T_{es} (≈0.3°C) occurred in all eight subjects. $\bar{T}_{sk}$ was higher during L than F in all experiments. During exercise, the T_{es} threshold for sweating was higher in L, with no change in the slope of $\dot{m}_s$ to T_{es} between menstrual cycle phases. This rightward shift in T_{es} averaged 0.53°C for all conditions studied. Temperature regulation in healthy women varies with menstrual cycle phase as the onset of sweating occurs at an elevated T_{es} threshold in the

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ABSTRACT

Thermoregulatory responses were studied in eight women during three separate experimental protocols in both the follicular (F) and luteal (L) phases of the menstrual cycle. Continuous measurements of esophageal temperature (T_{es}), mean skin temperature (T_{sk}), metabolism, and forearm sweating ($\dot{m}_s$) were made during all experiments. Study I evaluated 35 minutes of seated cycle exercise (60% $\dot{V}O_2$ peak, T_a = 35°C, T_{dp} = 14°C). Study II involved both passive heat exposure and seated cycle exercise (80% $\dot{V}O_2$ peak; T_a = 50.0°C, T_{dp} = 18.5°C) to elicit a 0.8°C increase in T_{es}. Study III evaluated high intensity exercise (35 minutes, 80% $\dot{V}O_2$ peak) at T_a = 35°C and T_{dp} = 10°C. The normal L increase in resting T_{es} (≈0.3°C) occurred in all eight subjects. T_{sk} was higher during L than F in all experiments. During exercise, the T_{es} threshold for sweating was higher in L, with no change in the slope of $\dot{m}_s$ to T_{es} between menstrual cycle phases. This rightward shift in T_{es} averaged 0.53°C for all conditions studied. Temperature regulation in healthy women varies with menstrual cycle phase as the onset of sweating occurs at an elevated T_{es} threshold in the luteal phase when resting core temperature is elevated.

Key Words: Exercise, Females, Sweating, Thermoregulation

The evaluation of human temperature regulation during the menstrual cycle is not novel (1,3,7,8,9,15,16,17,19), however, investigators have generally failed to evaluate their data in a manner consistent with current understanding of the control system for temperature regulation (5,14). Oftentimes, investigators explained the subject's response to a thermal stress by pre- and post-experimental variables, or from steady-state data during the thermal stress. Few investigators have evaluated transient thermoregulatory analyses, which are consistent with control system changes (16,17,27). We previously reported delayed thermoregulatory responses for heat loss in the luteal phase of the menstrual cycle during exercise (27). Hessemer and Bruck in an elegant evaluation of thermoregulatory control during both heat and cold exposure at rest, and exercise in a cool environment demonstrated a luteal phase delay in both heat loss and heat production mechanisms during the nighttime hours (16,17). Earlier investigations during passive heating indicated a delay in the onset of sweating in the luteal phase (3,15) with no apparent change in cutaneous opacity pulses, a crude index of skin blood flow (15). Other authors have generally discounted the alterations in thermoregulatory control as playing an insignificant role in performance, and these studies have been recently summarized (9).

The elevation in resting core temperature during the luteal phase of the human menstrual cycle may be a manifestation of the altered control of heat loss mechanisms, changes in the levels of the reproductive hormones (25), or increased interleukin-1 levels (6) any of which may defend the elevated temperature (3,16,17,27). In the present study, we have attempted to further characterize

alterations in local sweating responses in follicular and luteal phase experiments during the combination of exercise and environmental heat stress. These experiments should define the latency in effector onset for conditions which have not yet been reported.

METHODS

Eight healthy women (Table 1) volunteered to serve as subjects for the experiments following approval of the protocol by the local human use review board. Each reported having normal menstrual cycle as defined by a regular periodicity. Daily basal body temperature (BBT) was taken during the course of the study to verify that a normal luteal elevation in BBT had occurred (20). All subjects were familiarized with the experimental techniques before each study.

Three separate experimental protocols were conducted. Three subjects were tested in all three studies, one subject was tested in two protocols and four subjects were tested in only one protocol. At least four subjects participated in any one study. In all three studies, the subjects were studied in both the follicular (days 4-7) and luteal (days 18-24) phases of the menstrual cycle. The luteal experiments were always run during the luteal phase increase in core temperature (16,20). Serum levels of progesterone and estradiol were not assayed and deemed unnecessary as the critical factor for luteal phase experiments was an elevated resting core temperature. Furthermore, the luteal phase increase in core temperature has been related to elevated circulating progesterone levels (25).

Subjects wore cotton shorts, tee-shirt, shoes and socks for all exposures (Intrinsic clothing insulation of 0.15 clo), and had not eaten during the 8-hour period before an experiment. In all experiments esophageal (T_{es}) and mean skin ($\bar{T}_{sk}$) temperature, metabolism (M), local sweating from the arm and/or chest and whole body sweating rate from weight changes were measured.

Study I

Four experiments were conducted on each subject (#1,2,4,6) during the late Fall. Each subject was tested at 0400 and 1600 h during the follicular and luteal phase. The environmental temperature (T_a) was 35°C with an average ambient water vapor pressure (P_w) of 1.73 kP_a. Upon arriving at the laboratory, the subject inserted the esophageal catheter and adjusted it to heart level (verified by a peak steady temperature),and was instrumented for both mean skin temperature (eight site,23) Local sweating was assessed by a ventilated dew point sensor (13,21) placed on the volar aspect of the forearm. A venous catheter was placed in an arm vein for subsequent blood sampling. After a 25-minute rest period, metabolic heat production was estimated by open circuit spirometry and a blood sample taken (5 ml).

Exercise began at 60% peak aerobic power at 60 rpm and continued for 30 minutes. All temperatures, sweating rate and heart rate were measured continuously. At 25 minutes of exercise, metabolic heat production was measured and another blood sample drawn. Epinephrine (E) and norepinephrine (NE) concentrations were analyzed by a radioenzymatic technique (Cat-a-Kit, Upjohn Diagnostics, Kalamazoo, MI).

Study II

Four experiments were conducted during the winter on each subject (# 1,2,3,4,5). Two experiments were conducted in which the subject was passively heated (T_a = 50.4°C, P_w = 1.6 kP_a), one during the follicular phase (days 4-6) and one during the luteal phase (days 19-22) of the menstrual cycle. In the other two experiments, the subjects exercised at approximately 80% $\dot{V}O_2$ peak during both the follicular and luteal phase in the same environment as described above. All experiments began at 0700h.

A separate room from the environmental chamber was used for equilibration and instrumentation as described above. The ambient temperature of this room was adjusted so that each subject felt comfortable and averaged 28.8°C db, P_w = 7.7 kP_a. T_{es}, $\bar{T}_{sk}$ and local skin temperature ($T_{s,l}$) adjacent to dew point sensors from the chest and forearm were continuously recorded on an HP 9816 computer. The sensors were ventilated with ambient air from the chamber. These air flows were calibrated in situ at the end of the experiment. Sweating rates were calculated as described previously (13,21) and can be measured with an accuracy of 0.05 $mg \cdot cm^{-2} \cdot min^{-1}$ with this system. Total body sweating rate was evaluated from changes in body weight corrected for convective and evaporative heat loss from the respiratory tract. Metabolic heat production was estimated periodically during the passive experiments and continuously during the exercise experiments by open circuit spirometry. Plasma norepinephrine and epinephrine concentrations were determined by a radioenzymatic technique (Upjohn, Cat-a-Kit) in blood samples drawn at rest in a thermoneutral environment, and during T_{es} increments

(0.2°C) during both passive and active experiments. The experiment was terminated after T_{es} had increased some 0.8°C above initial values or the subject complained of headache, light-headedness or nausea. The average time of the passive heating experiments was 178 min. Sampling times were matched for T_{es} during passive and active experiments on the same subject during a given cycle phase.

During the exercise experiments, the subject began to exercise at approximately 80% $\dot{V}O_2$ peak within two minutes after entering the environmental chamber. The average time of the exercise experiments was 9 min. Blood samples were drawn at the same T_{es} as during the passive experiments for either the follicular or luteal phase.

Study III

Subjects 1, 2, 4, 5, 7 and 8 participated in exercise experiments at 80% $\dot{V}O_2$ peak in a T_a = 35°C, P_w = 1.4 kP_a environment in both the follicular and luteal phase of their menstrual cycle. These exercise experiments were of a longer duration (35 minutes at 80%) to further evaluate thermoregulatory effector function during the human menstrual cycle. Subjects were instrumented for core (T_{es}) and skin temperature ($\bar{T}_{sk}$) measurements and arm sweating as described above. After thermal equilibration, a 20-minute rest period followed by 35 minutes of exercise occurred.

Statistical Analyses

In all experiments a regression equation was calculated for each subject during the exercise transient phase for the $\dot{m}_s$ to T_{es} relationship. The data collected after the subject reached steady state were not included in the regression. The T_{es} thresholds (defined as T_{es} intercept) for initiation of thermal sweating were calculated from the regression equation at $\dot{m}_s = 0.06$ mg•cm^{2}•min^{-1} (2). An analysis of variance was performed using the individual slopes and thresholds of the $\dot{m}_s$ to T_{es} relationships. Analysis of variance routines were used to compare all variables at the time of the blood samples or during steady-state exercise or rest where appropriate. Tukey's test of critical difference was used when necessary. All differences in the RESULTS are reported at $P<0.05$, unless otherwise noted.

RESULTS

Study I

Mean temperature and metabolic data at rest and during steady-state exercise are shown in Table 2. The early morning resting T_{es} averaged 36.76°C in follicular phase and 37.06°C in the luteal phase; late afternoon temperatures averaged 37.17°C and 37.48°C, respectively. These circadian differences averaged 0.41°C and 0.42°C for the follicular and luteal phases during the early morning and late afternoon, respectively. Resting $\bar{T}_{sk}$ was higher in the afternoon in both cycle phases with luteal afternoon T_{sk} higher than follicular afternoon T_{sk}. Chest skin wettedness was significantly higher in the morning during both cycle phases

with no difference between cycle phase. The steady-state exercise data presented in the lower panel of Table 2 do not indicate any effect of either the menstrual cycle of the circadian period on these measured variables with the exception of a lower steady-state esophageal temperature in the early morning of the follicular phase. However, the esophageal temperature for the onset of arm sweating averaged 36.44°C and 37.18°C at 0400h and 37.11°C and 37.46°C at 1600h for the follicular and luteal phases, respectively. These data are graphically shown for a representative subject in Figure 1. The sensitivity, or slope of $\dot{m}_s$ to increasing T_{es}, was unchanged by cycle phase or circadian period and averaged 1.12 $mg \cdot min^{-1} \cdot {}^{o}C^{-1}$.

Study II

Mean measured variables for the five subjects during passive heating for both cycle phases are presented in Table 3. The normal resting increase in T_{es} during the luteal phase averaged 0.31°C. Resting norepinephrine concentration averaged 74% higher in luteal experiments. The luteal phase elevation in T_{es} was continued through passive heating. $\bar{T}_{sk}$ was significantly higher during luteal phase experiments. The higher initial NE values during the luteal phase continued through all levels of T_{es} during passive heating. Chest and arm local sweating did not vary in passive experiments between cycle phase It is interesting to note that none of the 5 subjects could complete the required 0.8°C rise in T_{es} during the luteal phase. All were removed from the heat complaining of dizziness, headache or nausea.

The thermoregulatory variables during severe exercise at 50°C are shown in Table 4. Again, the luteal phase elevation in resting T_{es} is carried through exercise. $\bar{T}_{sk}$ is higher in luteal phase experiments throughout exercise compared to follicular experiments. Both arm and chest sweating are unchanged by cycle phase during exercise.

There was no statistically significant difference in $\bar{T}_{sk}$ within a given cycle phase between passive and exercise experiments. However, both arm and chest sweating tended to be higher in passive experiments than exercise experiments at the same T_{es} in both the follicular and luteal phases.

The threshold of $\dot{m}_s$ to increasing T_{es} was shifted rightward in the luteal phase during exercise compared to follicular experiments. Specifically, the T_{es} onset averaged 36.91°C in follicular experiments and 37.54°C during luteal. Figure 2 illustrates these data in a representative subject during the intense exercise.

Study III

Both resting and exercise steady-state temperature data are shown in Table 5 for the 6 subjects. Again, resting T_{es} is shifted higher in the luteal experiments and this elevation is carried through exercise. There is a trend for $\bar{T}_{sk}$ to be higher during luteal, but no other variables differ with menstrual cycle phase. The significant rightward shift in T_{es} for sweating onset is demonstrated in Figure 3 for one subject.

Table 6 presents a summary of the control of thermoregulatory sweating in the three different studies conducted. The studies range from moderate to severe work and from moderately hot to severely hot ambient temperature. Three different times of the day are also represented. In all three protocols examined during exercise, a rightward shift in the T_{es} threshold for the onset of sweating occurs independently of changes in the sensitivity of sweating to increasing T_{es}.

The time at which regulatory sweating occurs after initiation of heat and/or exercise exposure is shown in Table 7 for the 3 experimental protocols run. Sweating is initiated at a delayed time in all luteal phase experiments. Additionally, sweating is delayed ($p=0.08$) in the afternoon experiments compared to the early morning experiments.

DISCUSSION

Delayed thermoregulatory sweating is consistently found in normally cycling (eumenorrheic) women during the luteal phase of the menstrual cycle (3,15,16,17,27) Our data clearly indicate a threshold temperature elevation for sweating difference resulting from the elevation in body temperature during the luteal phase. This rightward shift in T_c occurs at all periods of the circadian cycle studied from early morning through late afternoon and at all exercise intensities examined. This latency in effector onset has been documented by other investigators (3,4,15,16,17,27) in varied environmental conditions and exercise stresses. The parallel changes in sweating and cutaneous blood flow responses (16,17,27) and

earlier heat production (16) responses when appropriate, indicate a central alteration in the control of these thermal responses and a true defense of the thermoregulatory set-point (5,12,14).

Many investigators have calculated total heat storage during an exercise and/or heat challenge in different menstrual cycle phases. This type of analysis ignores thermoregulatory control and is insensitive to the menstrual cycle changes in sweating that we report. We have presented our resting and steady-state data for comparative purposes. However, at issue is the concept of thermoregulatory control, not whether an individual can perform a walking or cycling task during heat or exercise exposure. The overwelming evidence from the current study and those of Hessemer and Bruck (16,17), Haslag and Hertzman (15) and Bittel and Henane (3) demonstrates a menstrual cycle effect on thermoregulatory control during both exercise and passive thermal exposure. The slope of sweating to core temperature was not affected at any environmental temperature or exercise state in the present study which is in agreement with earlier work (3,4,15,27). However, other investigators have shown an increased sensitivity of one or more of the thermoregulatory responses during the luteal phase (16,17,18). In general, the slope of a thermoregulatory response is influenced by peripheral alterations at the effector, such as occurs with hypobaria (21) or exercise training (24). The inconsistent findings between the present evaluations and those of other investigators (16,17,18) is unexplained.

We have shown that mean skin temperature generally is elevated in the luteal phase at rest and during exercise. Haslag (15) demonstrated this same response in two of her three subjects, however Bittel (3) showed a slightly lower skin temperature in the luteal phase. Kenshalo (19), demonstrated that women defended a warmer skin temperature in the luteal phase, and in another study women perceived thermal comfort at a higher core temperature during the luteal phase when given a cutaneous thermal challenge (7). It appears that both a warmer core temperature and a warmer skin temperature may be defended during the luteal phase. Others (1,11,16,17,29,30) have not been able to demonstrate differences in skin temperature as a result of the menstrual cycle, however previous exposure or activity may have influenced experimental findings. Interestingly, resting plasma norepinephrine concentration is higher in the luteal phase in resting women at a comfortable temperature. This higher NE concentration is carried over to passive heat stress and exercise in the current studies. However, elevated levels of 17-β-estradiol in the luteal phase have been shown to inhibit norepinephrine binding to vascular smooth muscle(28). Therefore, the higher circulating levels may have no impact on the cutaneous vasculature. The higher skin temperature seen may be a result of the decreased norepinephrine binding at peripheral vascular sites with a resultant elevation in cutaneous blood flow and surface temperature. An elevation in skin temperature normally results in an earlier onset for heat loss responses, i.e. sweating and vasodilation (10,24). Paradoxically, sweating and vasodilatory onset are at a higher esophageal temperature when skin temperature is slightly higher. The skin influence appears to be overridden by the mechanism which altered thermoregulatory control.

Numerous studies have evaluated the effect of the menstrual cycle on temperature regulation, with the majority indicating negative results (1,9,11,29,30). That is, the menstrual cycle has no effect on the ability of a human female to regulate her body temperature during exercise or environmental heat stress. Wells et al. (29,30) evaluated both passive heating and exercise without finding any temperature differences in arbitrarily assigned (by cycle day) cycle phases. Unfortunately, these investigators did not demonstrate an elevated resting core temperature before subjecting these individuals to exercise or passive heat exposure (29,30). Similarly, Avellini et al. (1) failed to demonstrate differences in resting body temperature before exposure to humid heat and consequently failed to report a menstrual cycle effect on the temperature parameters measured.

Cunningham et al. (8), in a comparison of men and women during passive heat or cold exposure, demonstrated delayed effector onset in women compared to men. However, the women were studied at various times of the day, at varying phases of the menstrual cycle and were more that likely less fit than their male counterparts. An earlier study (24) involving both male and female subjects demonstrated differences in trained state and heat acclimation state on the onset and sensitivity of cutaneous blood flow and sweating responses without controlling for menstrual cycle phase. Bittel and Henane showed that the less fit female subjects of their study responded in a similar manner than the male subjects to heat stress although the females were less tolerant to the heat (3). The three studies discussed here evaluated changes in the control of temperature regulation during environmental stress, however each studied was confounded by the lack of

control of factors (trained state, cycle phase, circadian phase) known to affect the onset of heat loss mechanisms. Drinkwater (9) in a recent review concludes that in order to compare thermoregulatory responses of male and female subjects, it is required that all subjects exercise at the same relative percent of maximal aerobic power. However, in many of these studies (8,24) the investigators neglected to control for menstrual cycle phase, or arbitrarily tested individual subjects at pre-defined intervals that did not necessary correlate with an elevated body temperature (1,30,31).

The effect of the menstrual cycle on physiological responses of women to exercise and heat stress has been summarized recently by Drinkwater (9). Most of those studies concluded that performance is unaffected by the menstrual cycle. Performance, however, should not be confused with thermoregulation. The data from this study and from several other investigations (3,15,16,17,27) clearly indicate that the regulation of body temperature is affected by the menstrual cycle. Specifically, an elevated resting core temperature occurs in ovulatory women, which is defended by a delayed onset for those thermal mechanisms involved with heat loss. Bittel has suggested that performance is decreased due to the increased heat storage in the luteal phase (3), a point also mentioned by Hessemer (17) and supported by the passive heating data of the present study. Hessemer (16,17) conducted studies in the early morning hours to more fully take advantage of the the greatest difference in core temperature during the luteal and follicular phase. In the current set of studies, we have documented the delayed thermoregulatory response at many times of the day and can unequivocally claim a response. This

delay is apparent during passive heating at a high ambient temperature, during both moderate and high intensity exercise at a moderate environmental heat load, and during severe exercise in a high ambient temperature.

Problems associated with evaluating sweating to core temperature and cutaneous vasodilation to core temperature relationships, are greatly complicated when heat acclimation state (24), time of day (26) and state of physical training are variable in an experimental condition. The evaluation of pre to post responses or "steady-state" comparisons do not wholly show effects of homeostatic control of body temperature. Add to these factors the effect of elevated core temperature during the luteal phase of eumennorreic women, and more variability confounds the individual regression analysis and virtually absolves any comparison made of the regression analysis from subject to subject. The study of thermoregulatory control in human females is at best difficult since specific days for study when comparisons can be made are limited, time constraints are placed on subjects due to academic calendars, acclimation state, trained state, and changing reproductive function. However, if females are to be evaluated during thermal stress of endogenous or exogenous origin, all of the above conditions must be considered.

In summary, the present study provides concrete evidence of the latency in heat loss responses associated with the luteal phase of the human menstrual cycle. This investigation points to clear evidence that delayed thermoregulatory effector onset is a consistent finding during passive heating or cooling (16) as well as during exercise in hot environments at a number of different exercise intensities.

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Figure legends

Figure 1. Forearm sweating versus esophageal temperature for a representative subject during moderate cycle exercise in 35°C. Both follicular (•) and luteal (Δ) phase data are presented at 1600 h.

Figure 2. Forearm sweating versus esophageal temperature during intense exercise at 50°C for a representative subject. Follicular (•) and luteal (Δ) phase data are plotted during 0800 h experiments.

Figure 3. Forearm sweating versus esophageal temperature during moderate exercise in 35°C at 0800 h. The data presented are from a single subject in both the follicular (•) and luteal phases (Δ).

Table 1. Individual Subject Characteristics

Subject	Age (yr)	Height (cm)	Weight (kg)	A_D* (m^2)	$\dot{V}O_2$ peak$^{\tau}$ ($\ell \cdot min^{-1}$)
1	32	173.0	64.2	1.77	2.70
2	26	162.0	60.9	1.65	2.65
3	27	165.1	64.0	1.71	2.55
4	30	170.0	59.0	1.68	2.55
5	21	162.6	68.0	1.73	2.64
6	34	156.0	48.4	1.45	1.90
7	26	173.0	77.8	1.91	3.83
8	24	162.6	56.0	1.57	2.73
$\bar{X}$	27.6	165.5	62.3	1.68	2.69
S.D.	4.2	6.0	8.7	0.14	0.53

* DuBois surface area

$^{\tau}$ Modified ergometer, with subject seated in a contour chair behind the pedals, legs parallel to floor.

Table 2. Thermoregulatory variables at rest and during steady-state exercise T_a = 35°C (STUDY I).

	T_{es} (°C)	$\bar{T}_{sk}$ (°C)	$\dot{m}_s$ (mg•cm^{-2}•min^{-1})	M (W•m^{-2})	E_{sk} (g•min^{-1})	w (%)	NE (ng•ℓ$^{-1}$)	E (ng•ℓ$^{-1}$)
REST								
Foll. am	36.76	34.98	0.10	41	_	0.46	225	42
	(.10)	(.36)	(.10)	(7)		(.14)	(78)	(21)
Foll. pm	37.17*	35.83*	0.12	42	_	0.25*	252	76
	(.20)	(.37)	(.14)	(5)		(.14)	(95)	(18)
Lut. am	37.06	35.12	0.09	42	_	0.54	267	42
	(.13)	(.44)	(.05)	(3)		(.05)	(88)	(20)
Lut. pm	37.48*	36.34*	0.10	44	_	0.14*	302	76
	(.26)	(.41)	(.07)	(3)		(.10)	(103)	(48)
EXERCISE								
Foll. am	37.59	34.95	0.93	314	12.1	0.90	1178	137
	(.19)	(.54)	(.21)	(82)	(3.6)	(.10)	(509)	(78)
Foll. pm	38.01*	35.52	0.89	289	10.6	0.92	1139	137
	(.31)	(.24)	(.12)	(46)	(2.3)	(.18)	(628)	(51)
Lut. am	37.97*	35.01	0.87	297	10.8	0.94	1091	120
	(.23)	(1.06)	(.21)	(28)	(1.3)	(.05)	(341)	(48)
Lut. pm	38.01*	35.54	0.87	299	11.2	0.97	1201	129
	(.25)	(.63)	(.12)	(26)	(1.3)	(.14)	(723)	(39)

* different from Foll. am $p<0.05$.

Values are mean ± standard deviation for 4 subjects. T_{es}, esophageal temperature; $\bar{T}_{sk}$, mean weighted skin temperature; $\dot{m}_s$, arm sweating; M, metabolic heat production; E_{sk}, evaporative heat loss from the skin calculated from weight changes pre and post- exercise; w, chest skin wettedness; NE, plasma norepinephrine concentration; E, plasma epinephrine concentration

Table 3. Thermoregulatory variables during passive heating T_a = 50°C (STUDY II).

Time (min)	T_{es} (°C)	$\bar{T}_{sk}$ (°C)	M ($W \cdot m^{-2}$)	$\dot{m}_{sch}$ ($mg \cdot cm^{-2} \cdot min^{-1}$)	$\dot{m}_{sa}$	HR ($b \cdot min^{-1}$)	NE ($ng \cdot \ell^{-1}$)	E	E_{sk} ($g \cdot min^{-1}$)
Follicular									
Pre	36.97	-ξ	-	-	-	-	189	60	
	(0.23)						(105)	(12)	
30(6)	37.17	37.83	37	0.5	0.5	77	220	56	
	(0.21)	(0.45)	(3)	(0.4)	(0.4)	(16)	(43)	(29)	
65(19)	37.37	37.35	41	0.8	0.9	79	232	66	
	(0.23)	(0.19)	(7)	(0.4)	(0.5)	(13)	(56)	(17)	
116(40)	37.56	37.35	38	0.9	1.0	80	338	126	
	(0.23)	(0.33)	(3)	(0.5)	(0.4)	(14)	(143)	(29)	
165(141)	37.69	37.44	39	1.0	1.1	83	338	59	7.4
	(0.19)	(0.22)	(7)	(0.4)	(0.4)	(8)	(155)	(32)	(1.3)
Luteal									
Pre	37.28*	-	-	-	-	-	342*	98	
	(0.22)						(109)	(43)	
32(9)	37.48	37.96*	37	0.7	0.5	77	307*	73	
	(0.21)	(0.12)	(6)	(0.4)	(0.3)	(13)	(84)	(35)	
78(21)	37.67	37.43*	35	1.2	0.7	85	381*	127	
	(0.20)	(0.16)	(7)	(0.4)	(0.1)	(10)	(159)	(64)	
169(66)	37.84	37.75*	43	1.3	1.0	92	545*	183	6.5
	(0.16)	(0.38)	(5)	(0.4)	(0.2)	(8)	(114)	(76)	(1.2)
178(68)	-	-	-	-	-	-	-	-	

* different from follicular $p < 0.05$.

Values are mean ± standard deviation for subjects. T_{es}, esophageal temperature; T_{sk}, mean weighted skin temperature; $\dot{m}_{sch}$, chest sweating; $\dot{m}_{sa}$, arm sweating; NE, plasma norepinephrine concentration; E, plasma epinephrine; E_{sk}, evaporative heat loss from the skin, calculated from weight changes pre- and post-exposure.

ξ There are not sufficient data on all subjects for the mean data to be presented. The pre data was collected at T_a = 28.8°C, time listed is when blood samples were taken approximating 0.2°C rise in T_{es}.

Table 4. Thermoregulatory variables during exercise (80% $\dot{V}O_2$ peak), T_a = 50°C (Study III).

Time	T_{es}	$\bar{T}_{sk}$	M	$\dot{m}_{sch}$	$\dot{m}_{sa}$	HR	NE	E	E_{sk}
(min)	(°C)	(°C)	($W \cdot m^{-2}$)	($mg \cdot cm^{-2} \cdot min^{-1}$)		($b \cdot min^{-1}$)	($ng \cdot \ell^{-1}$)		($g \cdot min^{-1}$)
Follicular									
Pre	36.96	-ξ	-	-	-	-	225	92	
	(0.22)						(87)	(31)	
3.5(.9)	37.17	37.82	341	0.6	0.7	147	614$^{\tau}$	171	
	(0.21)	(0.40)	(30)	(.4)	(.4)	(10)	(230)	(73)	
4.6(1.1)	37.46	37.73	379	0.9	1.0	152	811$^{\tau}$	174	
	(0.29)	(0.42)	(60)	(.2)	(.4)	(16)	(463)	(118)	
7.0(1.0)	37.65	37.46	375	1.0	1.0	155	1107$^{\tau}$	235	
	(0.30)	(0.27)	(53)	(.3)	(.4)	(16)	(599)	(129)	
8.6(1.1)	37.78	37.32	395	1.1	1.1	166	1350$^{\tau}$	278	16.9
	(0.28)	(0.35)	(44)	(.4)	(.4)	(15)	(796)	(126)	(3.6)
Luteal									
Pre	37.21*	-	-	-	-	-	380*	177	
	(0.27)						(128)	(157)	
4.0(1.4)	37.47	38.51*	353	0.7	0.8	146	815$^{\tau}$	101	
	(0.22)	(0.38)	(22)	(.4)	(.2)	(4)	(670)	(50)	
5.6(1.8)	37.68	38.23*	377	1.1	1.0	158	865$^{\tau}$	213	
	(0.23)	(0.42)	(22)	(.4)	(.3)	(13)	(613)	(95)	
7.2(2.7)	37.83	37.03*	390	1.4	1.0	160	589$^{\tau}$	183	
	(0.24)	(0.39)	(11)	(.5)	(.3)	(11)	(206)	(82)	
8.4(2.9)	37.98	37.83*	397	1.4	1.1	165	919$^{\tau}$	246	17.2
	(0.25)	(0.38)	(26)	(.5)	(.4)	(11)	(308)	(123)	(2.8)

* Different from follicular $p < 0.05$, $^{\tau}$ different from passive $p < 0.05$.

Values are mean ± standard deviation for subjects. T_{es}, esophageal temperature; T_{sk}, mean weighted skin temperature; M, Metabolic heat production; $\dot{m}_{sch}$, chest sweating; $\dot{m}_{sa}$, arm sweating; HR, heart rate; NE, plasma norepinephrine concentration; E_{sk}, evaporative heat loss from the skin, from weight changes.

ξ These data were not collected in the pre exercise period. The pre data were collected at T_a = 28.8°C, time is when blood samples were taken approximating 0.2°C rise in T_{es}.

Table 5. Rest and steady-state exercise (80% $\dot{V}O_2$ peak) at T_a = 35°C (Study III).

	T_{es} (°C)	$\bar{T}_{sk}$ (°C)	M ($W \cdot m^{-2}$)	$\dot{m}_{sa}$ ($mg \cdot cm^{-2} \cdot min^{-1}$)	HR ($b \cdot min^{-1}$)	E_{sk} ($g \cdot min^{-1}$)
REST						
follicular	36.86 (.17)	35.81 (.26)	46 (9)	0.11 (.03)	67 (3)	-
luteal	37.13* (.17)	36.09 (.34)	46 (15)	0.07 (.01)	70 (4)	-
EXERCISE						
follicular	38.14 (.29)	35.14 (.62)	429 (45)	1.05 (.17)	156 (10)	17.0 (4.8)
luteal	38.39* (.31)	35.39 (.63)	445 (46)	0.95 (.24)	159 (7)	17.2 (5.4)

* different from follicular, $p < 0.05$.

Values are mean $\pm$ standard deviation for 6 subjects. T_{es}, esophageal temperature; T_{sk}, mean weighted skin temperature; M, metabolic heat production; $\dot{m}_{sa}$, arm sweating; HR, heart rate; E_{sk}, evaporative heat loss from the skin, calculated from weight changes pre- and post- exposure.

Table 6. T_{es} threshold (°C) for initiation of sweating and slope ($mg \cdot cm^{-2} \cdot min^{-1} \cdot K^{-1}$) of linear regression equation generated from transient response to exercise.

	Follicular		Luteal	
STUDY I				
	Threshold	Slope	Threshold	Slope
0400h	36.44 (.26)	0.93 (.15)	37.18* (.13)	1.33 (.53)
1600h	37.11# (.17)	1.05 (.17)	37.46*# (.43)	1.15 (.49)
STUDY II				
0800h	36.91 (.21)	1.48 (.17)	37.54* (.25)	1.33 (.50)
STUDY III				
0800h	36.66 (.23)	1.09 (.28)	37.08* (.20)	1.10 (.14)

* $p<0.05$ different from follicular
# $p<0.05$ different from 0400 h

Table 7. Onset time (minutes) for initiation of local sweating.

	Follicular	Luteal
STUDY I		
0400h	1.3 (.5)	3.0 (1.4)*
1600h	2.8 (1.7)	4.0 (1.4)*
STUDY II		
Passive	5.0 (2.9)	6.3 (6.5)
Exercise	1.6 (1.3)	2.2 (1.6)
STUDY III		
0800h	0.9 (.2)	2.0 (.9)*

* $p<0.05$ different from follicular

FIG. 1

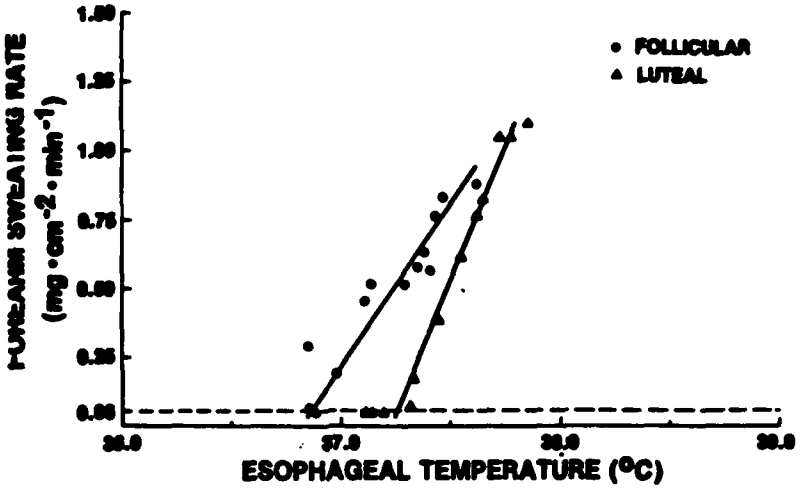


FIG. 2

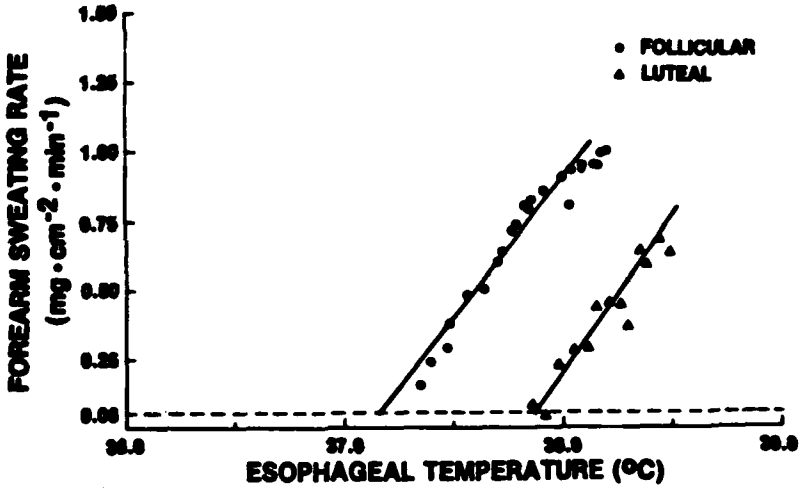
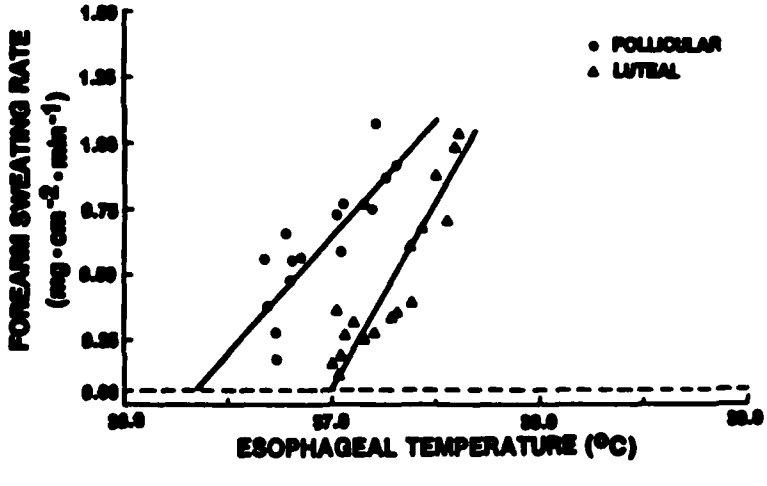


FIG. 3



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